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The Effect of Hydrocarbons on Tuberculo-allergy and Tuberculo-immunity.—Several investigators have reported the superior results obtained by inoculating dead tubercle bacilli in paraffin oils, as compared with those in saline, in producing allergic hypersensitiveness. A study of this phenomennon seemed worth while especially as it might throw some light on the specific allergic and immune reactions in tuberculosis. For this purpose varying amounts of nonviable (heat killed) and viable human tubercle bacilli were injected into guinea pigs after suspension in saline or paraffin oils. Before tubercle bacilli were suspended in paraffin or similar oils all extraneous water was removed by drying over phosphoric anhydride in a vacuum desiccator. This drying does not impair the viability of live tubercle bacilli. The effect of suspension in mineral oil on the growth of tubercle bacilli on media was studied, and also the effect of certain volatile paraffin hydrocarbons, instead of mineral oil, both on growth and immunity production. The authors' summary and conclusions follow: (*1*) While viable avirulent human tubercle bacilli sensitize guinea pigs allergically in quantitative tests grading down to 0.000,001 mg. in suspension in saline solution, mineral oil suspensions abruptly fail in sensitizing with amounts of less than 0.01 mg. Heat killed bacilli in saline also fail to sensitize in amounts below 1.0 mg., but the difference noted is the gradual diminution of sensitizing power in saline as compared with the abrupt discontinuance with mineral oil. This effect with mineral oil suspension of tubercle bacilli also is displayed in growth limit (culture) and infection (guinea pig) experiments. (*2*) The spread of viable avirulent human tubercle bacilli from the site of subcutaneous inoculation, using cultures from the spleen as an index, is slightly greater when the bacilli are suspended in mineral oil as compared with saline suspensions. (*3*) With small amounts of viable avirulent human tubercle bacilli, the route of injection determines perceptibly the lethal effect of general tuberculo-allergic intoxication (produced by natural filtrate): the intravenous route proves about 100 times as effective. The same factor appears to be instrumental when heat killed tubercle bacilli are given intravenously and compared with the subcutaneous route. Heat killed tubercle bacilli suspended in mineral oil and injected subcutaneously prove much more efficient for sensitizing to lethal general allergy than when the same bacilli are suspended in saline. (*4*) When dry, tubercle bacilli are extremely resistant to heat, surviving 120°C. for 15 minutes; while in saline suspension, they are killed at 65°C. almost instantly. (*5*) Storage of human tubercle bacilli at incubator temperature (37°C.) in suspension in mineral oil and the volatile hydrocarbons, octane and decane, results in rapid loss of viability of the bacilli (within 2 to 4 days). A retardation of this effect occurs at refrigerator temperature in comparison. (*6*) Mineral oil or decane suspensions of non-viable tubercle bacilli are far less efficient specific tuberculo-immunizing agents than are saline suspensions of viable avirulent tubercle bacilli. However, mineral oil suspensions of non-viable tubercle bacilli produce a relatively high grade allergic sensitivity when given subcutaneously, but this

effect is not appreciable when the bacilli are suspended in decane. The tuberculo-allergic sensitivity is no measure of the grade of the existing specific immunity. (7) From the foregoing experiments, it would appear that there are two main factors in producing the accentuated tuberculo-allergic sensitization when mineral oil suspensions of non-viable tubercle bacilli are injected subcutaneously (as compared with saline suspensions). A. The modified absorption and reaction to the heat killed tubercle bacilli by the body as a result of the interplay of the mineral oil. B. The apparent accelerated spread of the bacilli in the body when suspended in mineral oil—and this appears less important. The entire effect appears to be not by the mineral oil or hydrocarbons on the tubercle bacilli per se but rather on the body's reaction to these mixtures.—*The Effect of Paraffin Hydrocarbons on Tuberculo-allergy and Tuberculo-immunity Produced by Tubercle Bacilli, H. J. Corper & M. L. Cohn, Am. J. Clin. Path., February, 1942, 12: 73 (Authors' abstract).*—(*J. S. W.*)

Respiratory Function of the Digestive Tract.—The only place of physiological obstruction in the lower portion of the oesophagus is at the loop-like para-oesophageal muscle of the diaphragm which contracts and relaxes synchronously with the respiratory movements of the diaphragm. Air enters the stomach and intestines of the newborn and adults during the inspiratory movements of the thorax. The amount of air which can be swallowed has no substantial significance either in adults or in infants. The term "aerophagy," which does not correspond to the actual mechanism of accumulation of air in the stomach, should be replaced by a more correct term, namely, that of "gastrospiry." At present there is sufficient evidence to consider it well established that, in addition to pulmonary respiration, every human has a gastric respiration which may occasionally be rather marked. The presence of air in the digestive tract of a newborn can serve as proof of extra-uterine respiration and hence as proof that the infant was born alive. The roentgen examination of a stillborn infant at any time of its uterine life never reveals any traces of air in the digestive tract. Correctly made roentgenograms of dead foetus that were breathing even for a very short time always disclose the presence of air in the stomach or intestines, no matter how small its quantity may be. Therefore such a roentgenogram serves as a test of life.—*The Respiratory Function of the Digestive Tract as the Basis of the Roentgenographic Life Test, J. G. Dillon, Am. J. Roentgenol., November, 1942, 48: 613.*—(*W. H. M.*)

Anatomy of the Bronchial Tree.—The form of the bronchial tree and its distribution are described in considerable detail. From a consideration of the embryology and comparative anatomy of the bronchial tree, it is shown that the fundamental design in each lung consists of a stem bronchus having a series of dorsal and ventral branches. The upper part of the lung has a separate stem with similar branches; on the right this stem, the eparterial bronchus, has an independent origin, while on the left it develops from a special ascending branch of the first ventral hyparterial bronchus. After reviewing accounts of the anatomy of the bronchi in man, a description of the human bronchial tree is given, based upon a study of 200 bronchograms as well as dissections, inflation preparations and celloidin casts. Each important branch of the bronchial tree is described, together with its bronchographic appearance and the pulmonary segment which it supplies. In the nomenclature of the bronchial tree, it is suggested that the word "lobe" should be omitted when naming individual bronchi since the lobar distribution differs on the two sides. Thus the bronchi to the right and left upper lobes are distinguished as eparterial and hyparterial, respectively, while the bronchus to the right middle lobe and its equivalent on the left side are called the right and middle bronchi; the bronchus to the lower lobe is termed simply the lower bronchus. In over 80 per cent of subjects the eparterial bronchus

has three branches—apical, anterolateral and posterolateral; the lateral or axillary branch with its separate segment, formerly described as common, is in fact a rare variation. The importance of this concerns the localization and bronchial drainage of lesions in the right upper lobe; for the axillary area is actually drained by branches of the anterolateral and posterolateral divisions; on looking into the eparterial bronchus, no branch going exclusively to the axilla is usually visible. Other modes of branching of the eparterial bronchus, and those affecting the hyparterial bronchus on the left side, are also described. The arrangement of the bronchi in the rest of the lungs was found to be substantially that described by most previous authors.—*The Anatomy of the Bronchial Tree, A. F. Foster-Carter, Brit. J. Tuberc., January, 1942, 36: 19.*—(*E. H. R.*)

Roentgen Diagnosis of Pneumonoconiosis and Silicosis.—Experimental investigations have shown that most nonsiliceous dusts exert no appreciable effects on connective tissues, the reaction is not progressive and does not terminate in a fibrosis appreciable on gross examination. It would seem, therefore, that if abnormal shadows are observed in roentgenograms of individuals exposed only to nonsiliceous dusts some other condition is present. There are two conditions which can be diagnosed or suspected because of their roentgenographic appearances. In baritosis, a form of pneumonoconiosis found in the baryta mining industry of Italy and in a few workers in a Pennsylvania plant, the lungs are studded with small, dense, sharply circumscribed nodules produced by the collection of mineral dust unaccompanied by fibrous tissue overgrowth. The less dense pseudonodulation occurring in arc welders reported by Enzor and Sander was due to the collection of iron particles without fibrous tissue reaction. The roentgen observations in simple silicosis may be summarized as follows: The shadows of the trachea, heart and aorta are normal. The shadow and movement of the domes of the diaphragm are not affected unless a coincident emphysema is present. The shadows of the hila may only occasionally seem large. Slight to moderate variations in the trunk shadows and linear markings are difficult to evaluate. The shadows of nodulation (2 to 6 mm. in size) are usually bilateral in distribution but variations may occur, such as lobar or unilateral distribution. The shadows of the nodules may be difficult to differentiate from those occurring in some types of tuberculosis, metastatic malignancy and fungus infestation, etc. Not all silicotic individuals develop generalized nodulation detectable on the roentgenogram. Conglomerate lesions result from a combination or coalescence of discrete nodules or may occur as such primarily. It is possible that the conglomeration may have occurred because the portion of the lung in question was previously damaged by a localized inflammatory process occurring before or during the early period of the dust exposure. Their shadows are usually found in the upper half of the lung fields and are difficult to evaluate in a single examination. Superimposed metastatic malignancy and lesions due to an active infection may produce similar appearances. Hyperventilation and emphysema may be present. All cases of silicosis with detectable evidence of infection are included in the next major group, silicosis with infection. Probably in the majority of instances, the superimposed infection is tuberculosis. The various roentgen findings may be summarized as follows: The trachea may be in the midline or displaced depending on the degree and direction of the traction by the fibrosis. Cor pulmonale is occasionally encountered in advanced silicosis but some consider it coincidental. The heart and aorta may be displaced by adhesions. If considerable emphysema is present the domes of the diaphragm may be depressed and limited in their movements and individualization of their costal components will be quite marked. The hila may be displaced by adhesions. The evaluation of trunk shadows and linear markings rarely help in arriving at a diagnosis. Bronchiectasis is difficult to detect in many instances. The pulmonary changes consist

of mottling and nodulation. Mottling refers to the small and poorly defined shadows produced by infection, usually as a result of a bronchogenic spread of a tuberculous lesion or the result of a fungus infestation. The shadows of soft nodulation, corresponding to the perinodular cellular reaction observed by the pathologist, are much larger than those produced by nodulation and mottling, but smaller than the conglomerate shadow. These shadows are much more likely to change in character in subsequent examinations than is the shadow produced by nodules, and change less than those due to mottling. The shadows of nodulation are similar to those described under simple silicosis. Soft nodulation cannot be demonstrated by a roentgen examination in every case. The shadows of the massive lesions vary from 3 to 20 cm. in size and are variable in shape. These lesions are usually due to extensive areas of fibrosis and are found in the upper third or two-thirds of the lung field. Formerly most of these lesions were thought to be due to silicosis with infection. This is now questioned. Gardner thinks that a third essential factor may be minerals other than free silica. Occasionally, massive lesions are simulated by necrotic, anemic infarcts or cavities in which there is no microscopical evidence of infection. Tuberculous cavities, usually thick-walled, may occur in the upper or lower lobes. Emphysema is frequently noted in the bases, along the periphery of the lobes and in the apices in cases of silicosis with infection. Bleb formation is noted particularly peripheral to the shadows of massive lesions. In some cases of spontaneous pneumothorax, excessive respiratory embarrassment is manifest. All the roentgen manifestations described take into consideration the hypothesis that the lesions have been produced by free silica and modified by other dusts. One should therefore bear in mind that the manifestations of silicosis in the various industries are likely to be different. The roentgen findings may differ even in different foundries. In most instances it requires many years (six to fifteen) of exposure before one can demonstrate roentgen evidence of silicosis. The exceptions to this rule are classified under the heading of "rapidly developing silicosis." In the advanced cases, lobar consolidations are due largely to superimposed infection obscuring changes through the silicosis. In earlier cases, the roentgenographic manifestations are those observed in interstitial pneumonitis. Most of the fatal cases have died with tuberculosis.—*Some Considerations Concerning the Roentgen Diagnosis of Pneumoconiosis and Silicosis, E. P. Pendergrass, Am. J. Roentgenol., November, 1942, 48: 571.*—(*W. H. M.*)

Workmen's Compensation and Silicosis.—Although silicosis was described in Switzerland as early as 1894, and again at various intervals, the list of industrial poisons compiled in 1911 did not contain silica. Consequently no provision for compensation of workers suffering with silicosis existed. In 1930, two cases of fatal silicosis were reported to the Swiss Workmen's Compensation Board with the claim that the families be compensated. The list of industrial poisons was supplemented from time to time, but in 1930 it was uncertain whether only free silica or both it and the silicates were responsible for the development of the disease. From then on an increasing number of reported cases caused the compensation board to voluntarily take the responsibility for the disease until such a time as the law could be amended. In 1938, silica was finally added to the list of industrial poisons, and workers now had a claim for compensation, whereas previously they were subject to the whim of the board. Between the years 1932 to 1941, a total of 428 silicotics received compensation. Of this number, 37 per cent were dead, 31 per cent unable to work, 5 per cent under treatment, and 27 per cent able to work. The participation of the different industries varies. Numerically the miners are in the first place, but the disease is most serious in sand blasters. When the figures are broken up, it is apparent that 51 per cent of the sandblasters have died, whereas only 38 per cent of the miners, and of the cases occur-

ring in workers in the ceramic industry only 13 per cent have died. The compensation board is entitled to curtail the pension if the disability was not wholly or principally caused by the industrial poison. Complications such as myocardial disease, alcoholism, bronchiectasis and tumors fall into this group. Superimposed tuberculosis is frequent, and the case is fully compensable, but cases of tuberculosis without evidence of silicosis, who present claims simply because of employment in a dust industry, are disallowed. Complications which must be compensated in full are bronchopneumonias, gangrene of the lung, spontaneous pneumothorax, right heart disease and perforation of a traction diverticulum of the oesophagus. Although the law provides that patients should be treated, little can be done for them. If the disease is far advanced, treatment resolves itself into treatment of the complications. In moderate cases, short sojourns in sanatoria and breathing exercises are ordered. The law also orders the institution of prophylactic measures, both medical and technical. Periodic examinations aim at early diagnosis, and such patients are immediately excluded from the dusty occupation. Technically, the substitution of harmless materials instead of the offending one has been tried wherever possible. Measures to combat the development of dust are instituted, and the wearing of respiratory masks is being made compulsory. —*Silikose und schweizerisches Unfallversicherungsgesetz, F. Lang, Schweiz. med. Wchnschr., January 16, 1943, 73: 65.*—(*H. M.*)

Pneumonitis and Pericarditis.—A case of acute pneumonitis with electrocardiographic findings is reported. During the course of his illness the patient exhibited five distinct areas of pneumonitis. Simultaneously electrocardiographic changes consisting of elevation of the ST segment and T wave inversion, frequently with coving, were noted. The patterns changed rapidly and returned to normal too quickly to correspond with that due to myocardial infarction. The interpretation given to these electrocardiographic changes was that of acute pericarditis. Clinically, there was at no time any physical signs of pericarditis. Similar cardiographic changes were noted subsequently in 11 out of 100 cases of respiratory infection who had temperatures of 100°F. or over. It seems advisable to take electrocardiograms in severe cases of acute atypical pulmonary infection so that the diagnosis of acute pericarditis may be made early.—*Acute Pericarditis and Pneumonitis, C. C. Fuller & J. W. Quinlon, New England J. Med., September 2, 1943, 229: 399.*—(*J. B.*)

Pneumonia in Children.—The establishment of an aetiological diagnosis in infants suffering from pneumonia is particularly difficult because of the problem of obtaining proper materials for culture. The streptococcus produces a pneumonia characterized by a rapidly advancing severe illness accompanied by rapid accumulation of fluid in the chest and by extreme toxaemia. It usually follows virus infections, such as measles, pertussis and after other streptococcal infections, like scarlet fever and septic sore throat. Staphylococcal pneumonias occur usually after operations or influenza, have an insidious onset and later a purulent or seropurulent sputum from a severe tracheobronchitis and multiple cavities in the lung. Lung abscesses tend to break into the pleural cavity in young children. The influenza bacillus produces a haemorrhagic bronchopneumonia, particularly during epidemics of clinical influenza. Of the nonbacterial agents, the commonest is the virus of influenza, which in turn predisposes to severe bacterial infections. Virus infection of the lungs may also be produced by contact with exotic birds (psittacosis) or certain domestic birds such as the pigeon (ornithosis). Rickettsial diseases such as American Q fever and epidemic typhus fever produce similar pneumonias. Not every pneumonia which fails to respond immediately to sulfonamides is due to a virus infection. Even the common bacteria are sometimes resistant to sulfonamide therapy. Also the streptococcal and staphylococcal pneumonias require pro-

longed and intensive treatment with sulfonamides before responding.—*The Diagnosis of Virus and Bacterial Pneumonia in Children, M. Finland, New England J. Med., July 29, 1943, 229: 199.*—(*J. B.*)

Cold Agglutinins in Atypical Pneumonia.—Cold agglutinins are substances present in many normal sera. They are capable of clumping erythrocytes and are called "cold agglutinins" because their action appears usually at low temperatures only and is dispelled by warming to 37°C. Some of these cold agglutinins are specifically concerned with subgroups of type A blood, whereas others are nonspecific and clump red cells irrespective of group. They have also been called "auto-agglutinins" because they affect the cells of the individual in whose serum they appear. Normally they exist in small amounts capable of producing only a delicate reaction which disappears on dilution beyond 1:10 or 1:20. Auto-agglutinins of high degree occur rarely and without regularity in a number of diseases. However, in two known human diseases, paroxysmal haemoglobinuria and trypanosomiasis, auto-agglutinins acting at low temperature upon homologous erythrocytes are found with sufficient uniformity to be used as a diagnostic aid. The titre of cold agglutinins acting upon human erythrocytes of group 0 was measured in 219 patients with various diseases. In normal persons, the titre of cold agglutinins rarely exceeds 1:16. A regular increase above this level was found only in atypical pneumonias and in mumps complicated by orchitis. Of 83 unselected cases of atypical pneumonia, 44 possessed a titre of at least 1:32 and 23 (28 per cent) exhibited a titre of 1:128 or beyond. Further analysis of 37 cases of classic atypical pneumonias indicated that the titre usually begins to rise precipitously at the end of the first week of the disease and reaches a peak between the tenth and twentieth day from the onset of respiratory symptoms. This manner of appearance is similar to that of a specific antibody. Return to normal may not occur for a few months. Three cases with carefully accepted diagnosis of atypical pneumonia failed to show an increased titre. Other common bacterial infections of the respiratory tract such as a common cold, bronchopneumonia, tuberculosis, lobar pneumonia and influenza failed to influence the titre of the cold agglutinins in the blood. The use of this test as an aid in differential diagnosis is implied.—*Relation of Cold Agglutinins to Atypical Pneumonia, J. C. Turner, S. Nisnewitz, Elizabeth B. Jackson & R. Berney, Lancet, June 19, 1943, 1: 765.*—(*J. B.*)

Pulmonary Complications of Common Cold and Sinusitis.—In the course of mass miniature radiography in the Royal Air Force transient pulmonary shadows were noted in a high proportion of cases with symptoms of a common cold. One hundred of these cases were followed up clinically and radiologically until the lung fields were clear. Ninety-one recently had or were having a "cold in the head" characterized by a stuffy or running nose usually followed in a few days by a cough accompanied by a small amount of mucoid sputum. Only 9 denied a cold, cough or sputum. The duration of symptoms before radiographic study was most often seven to fourteen days. A few cases dated symptoms back as long as six weeks. Fever was only noticed in 11 cases. There was generally no relation between the duration of symptoms and the type and extent of the pulmonary lesion. Radiologically, the lesions were usually small, and usually, though not exclusively, in the lower lung fields, unilateral or bilateral (21 per cent). The distribution of lesions was what one would expect from a descending infection, the right lower lung field being most frequently involved. The lesions were largest within two or three days of the appearance of the upper respiratory infection. Spontaneous clearance was usually complete within two or three weeks. When of longer duration, a continued nasal infection in the form of sinusitis was always found. The radiological lesions fell into three main groups—bronchpneumonic, pneumonic and basal striations. The importance of chronic infections of the upper respir-

atory tract in the production of bronchiectasis can be inferred from these findings.—*Pulmonary Complications of the Common Cold and Sinusitis, J. A. Kennedy, Lancet, January 19, 1943, 1: 769.*—(*J. B.*)

Sulfonamides in Respiratory Diseases in Children.—The indication for the use of a sulfonamide drug is an acute bacterial infection of the respiratory tract that is obviously spreading and is a serious or potentially serious one. The dosage should be adequate, approximately one grain per pound per day until the temperature subsides. Thereafter cut the dosage in half and keep up a few days more until the infection is under control. The drugs of choice are sulfadiazine or sulfathiazole.—*The Use of Sulfonamides in the Treatment of Respiratory Infections in Children, C. A. Janeway, New England J. Med., July 29, 1943, 229: 201.*—(*J. B.*)

Blast Injury.—In the first World War and in the Spanish Civil War deaths without any signs of external injury occurring close to the site of detonation were observed. Barcroft and Zuckerman found in autopsies of animals placed near an explosion pulmonary haemorrhages. In the present war several reports on blast injury to the lungs have appeared. This paper deals with observations made in Malta from December, 1941 to April, 1942. Three mechanisms have been suggested to explain the pulmonary lesions in blast injury: (*1*) a suction wave following the blast, leading to rupture of the alveolar capillaries; (*2*) sudden distension of the lungs with air causing rupture of the lung tissues; (*3*) traumatic effect of the blast wave upon the chest wall. This last explanation seems most probable because of the fact that lung haemorrhages were seen to be more severe on the side nearest the explosion. Congestion of vessels and haemorrhages were also found in abdomen, brain and cord. Clinical findings are: perforation of ear drums with or without haemorrhage; diminished hearing; normal or elevated blood pressure; bloody sputum; pneumonia, starting forty-eight to seventy-two hours after the incident; abdominal rigidity; nausea; anorexia. No definite neurological signs were seen. Radiological examination reveals loss of translucency and congestion in the central and lower portions of the lung fields. The treatment consists of rest, heat, morphine, oxygen inhalation. The onset of serious symptoms can be delayed for forty-eight hours or more. In severe cases of blast injury death occurs within a few hours to forty-eight hours after the incident. The cause of death is unknown.—*The Pathological and Clinical Findings in Blast Injury, R. E. Turnbridge & J. V. Wilson, Quart. J. Med., July, 1943, 12: 169.*—(*G. C. L.*)

Mitral Stenosis Simulating Tuberculosis.—Chronic passive congestion of the lung, as seen in long standing cases of mitral stenosis, may lead to small calcific foci which may simulate old haematogenous tuberculosis. Such patients may also be subject to attacks of acute pulmonary oedema and the resultant increase in hilar shadows and perihilar consolidations may be taken for tuberculous infiltrations, especially if they occur in conjunction with such calcific foci. Two cases are reported, and in one the picture could be produced by having the man exceed his exercise tolerance to the point of dyspnoea.—*Vortäuschung von Lungentuberkulose durch akute und chronische Stauung bei Fällen von Mitralstenose, A. Adler, Schweiz. med. Wchnschr., May 1, 1943, 73: 530.*—(*H. M.*)

Actinomycosis.—A ten year old boy was cured of actinomycosis of the lung by the administration of 125 g. of sulfanilamid and 183 g. of sulfamidothiazol over a three months' period. The patient took ill with dry pleurisy, and after five months of ill health effusion developed. This persisted until it turned purulent four months later, and after another two months an *empyema necessitatis* developed which contained the ray fungus. The patient received sulfanilamid, 3 g. daily in ten doses, for one and one half months, and after this time the bacteriological findings improved. He then received "cibazol," 3

g. daily in six doses, and shortly thereafter the fungus disappeared entirely and the general condition improved. The fistula closed. An earlier course of "cibazol" had to be discontinued because of the appearance of drug rash and fever. The patient has remained well over an observation period of six months after the conclusion of treatment. —*Ein Fall von Lungenaktinomykose im fistulösen Stadium, geheilt durch protrahierte Sulfonamidtherapie, G. Fanconi, Schweiz. med. Wchnschr., February 13, 1943, 73: 175.*—(*H. M.*)

Treatment of Bronchial Asthma.—The authors analyze the results of treatment of 459 cases of bronchial asthma under observation from one to twenty years. In 207 cases reported seven years previously, almost all patients who were entirely symptom free in 1935 were still in excellent condition. Nineteen of 104 patients previously reported as improved are not 100 per cent relieved. Those unimproved in 1935 were for the most part still unimproved or dead. In the present study the patients were classified as "paroxysmal" or "chronic". The paroxysmal cases occur chiefly in early life and totalled 298 patients, as compared with 161 with chronic asthma. Ninety-three of the 298 paroxysmal cases obtained 100 per cent relief from symptoms as compared to only 4 of the 161 patients with chronic asthma. The percentage of improvement was also considerably higher in the paroxysmal group. Death occurred in 23 per cent of the chronic cases as compared to only 3.7 per cent in the paroxysmal group. Of the total of 48 deaths, asthma was the sole cause in 21 cases and a contributory factor in 16 instances. Morphine was known to have been given prior to death in 6 cases and probably caused death by preventing the expulsion of obstructing mucous plugs. In treatment the best results are obtained in those who have paroxysmal asthma caused by some allergen which can be found and eliminated. Symptoms in the successful cases are apt to begin in the first decade of life. Paroxysmal cases are uncommon in the age group past fifty. The prognosis is especially good for paroxysmal asthma in the age group twenty to twenty-nine. To prevent chronic asthma, with its relatively poor prognosis, preventive measures should be instituted in the children of allergic parents, and an allergic survey should be made at once if symptoms begin.—*Treatment of Bronchial Asthma: A Survey of the Value of Treatment in Four Hundred and Fifty-Nine Cases during Twenty Years, L. Unger & A. A. Wolf, J. A. M. A., January 30, 1943, 121: 325.*—(*G. L. L.*)

Roentgen Diagnosis and Treatment of Primary Pulmonary Neoplasms.—A diagnosis of primary lung tumor was made in 363 cases seen at the Massachusetts General Hospital in Boston during the ten-year period ending in April, 1940. Carcinoma was demonstrated histologically in 155 cases. Ninety cases (58 per cent) were epidermoid carcinoma; 23 cases (14.5 per cent) were adenocarcinoma; 13 cases (8.4 per cent) were undifferentiated carcinoma; 29 cases (18.7 per cent) were "oat cell" carcinoma. Eighty per cent of the patients were males. There were no females in the "oat cell" group. The early diagnosis of pulmonary tumors is largely a roentgenological problem. The findings are primarily the result of varying degrees of bronchial obstruction in most cases, a noticeable exception being the rounded or lobulated masses seen at a considerable distance from the hilum when the tumor arises in one of the smaller bronchi. In the benign adenomata, which constitute about 10 per cent of the entire group, rounded and sometimes pedunculated tumors were frequently demonstrated in the bronchial lumen by using the Potter-Bucky diaphragm, spot-films or planigraph. Furthermore, there was little or no enlargement of hilar lymph nodes, no invasion of surrounding lung tissue other than that simulated by occasional secondary inflammatory changes. The location of the benign adenomata was usually in a main stem bronchus. The outstanding characteristics of the adenocarcinoma group were the roentgen finding of a dense, rounded or lobulated mass close to the affected

bronchus or clinical evidence of metastasis in the brain or other distant organ. Epidermoid carcinoma has no characteristic roentgen findings. The roentgen appearance of "oat cell" and undifferentiated carcinoma is that of a rapidly growing tumor which infiltrates surrounding organs. Only 27 of the 155 cases, with histological proof of carcinoma, were considered operable at the time the diagnosis was made; 15 died following operation before leaving the hospital; 5 are living and well, apparently free from disease. Pneumonectomy in advanced cases carries an operative mortality of approximately 50 per cent. Lobectomy, which may be used successfully in early cases, has a low operative mortality of about 5 per cent. End-results of surgical treatment in the benign adenoma group is 89.2 per cent living and well, while in the group classified as malignant adenoma, the survival was only 8.7 per cent. Surgery offers the best chance for a five-year cure, and is advocated if the disease is discovered early. On this basis cases selected for roentgen therapy will be those who have already passed the curable stage or for some other reason are considered inoperable. The treatment in the great majority is therefore palliative. Small doses of properly applied roentgen therapy may relieve pain even in far advanced cases. Rather large doses up to 5,000 or 6,000 r given through a small field carefully centered over the primary tumor will often relieve the bronchial obstruction and establish drainage, reducing cough and fever. In no case is it justifiable to heavily irradiate large areas of infected lung tissue. It is of some importance to determine the type of tumor present since the treatment selected is to a considerable extent dependent upon the histopathology of the tumor. The benign adenomata, which are radioresistant, are nearly all curable by surgical means. Although epidermoid carcinomas are radioresistant, excellent palliative results have been obtained when the roentgen therapy was directed toward relief of the bronchial obstruction. "Oat-cell" and undifferentiated carcinomata are radiosensitive and usually disappear rapidly, producing excellent but only temporary results. There are no cures in this group and roentgen therapy is preferable to surgery.—*Roentgen Diagnosis and Treatment of Primary Pulmonary Neoplasms, G. W. Holmes, Am. J. Roentgenol., October, 1942, 48: 425.*—(*W. H. M.*)

Primary Intrathoracic Tumors.—The incidence of primary intrathoracic tumors is apparently increasing, and because with early recognition the majority can be cured by radical surgery, the diagnosis and management of this group is becoming very important. Primary tumors of clinical importance consist of bronchial adenomas (10 per cent) and bronchogenic carcinomas (90 per cent). The symptoms of both are mainly bronchial irritative and bronchial obstructive. Bronchiectasis is frequently seen in adenoma of the bronchus, but rarely in carcinoma. Pain is a late symptom in carcinoma. The diagnosis can be made in 75 per cent by bronchoscopy. The roentgenologist takes up where the bronchoscopist leaves off in the diagnosis of the peripheral types of carcinoma. The death rate will be decreased by surgical removal only with early diagnosis. Immediate attention to two factors is important: prompt bronchoscopy in all cases of protracted cough, expectoration and haemoptysis, and prompt surgical exploration in all cases of solitary enlarging pulmonary shadows not otherwise explained. Primary tumors of the mediastinum make themselves known by pressure on mediastinal organs. Neuralgia in shoulder or arm is always suggestive. Dermoids and teratomata are the most common benign tumors of the anterior mediastinum. The most common benign tumor of the posterior mediastinum is neurogenic in origin. Pain is early, symptoms of obstruction come late. The most common malignant mediastinal tumors are lymphosarcomata. They are extremely radiosensitive, in contrast to the radioresistance of benign tumors. Lymphosarcomata are inoperable when symptomatic. Benign tumors should be removed surgically, as they may later prove fatal by obstruction or malig-

nant degeneration. Mediastinal lipomata are very liable to become liposarcomata, the latter being extremely invasive. Finger-like processes may burrow along fascial planes to become palpable on the chest wall. Even with painstaking dissection, bits of fatty sarcomatous tissue may be left behind, with resulting recurrence. Because of their fluid consistency, the complete transmission of the cardiac impulse may result in a mistaken diagnosis of aneurysm. The character of a neurogenic tumor depends on whether it is derived from the myelin sheath, the sheath of Schwann or the connective tissue sheath. In the malignant group, neuroblastomata and neurosarcomata, erosion of ribs or of vertebral bodies is of grave diagnostic import. Radio-therapy is particularly valuable in this group. Benign tumors of neurogenic origin occurring in or near a spinal foramen may extend into the spinal canal, compressing the cord. The most interesting group of thymic tumors is that associated with myasthenia gravis. The majority of such tumors is benign. Malignant tumors of the thymus are not common and are invariably inoperable when symptoms have developed. The differential diagnosis of Hodgkin's disease from other mediastinal lymphomas is usually difficult. It is unusual for Hodgkin's disease to remain confined entirely to the mediastinum, and biopsy of a cervical or axillary node solves the problem. Multiple shadows confined entirely to the mediastinum are more likely to be sarcomatous in nature. Surgery offers nothing in either condition, but fortunately these lymphatic malignancies are very radiosensitive, although permanent cure seldom results. Superior sulcus tumors represent a variety of new growths arising in the superior paramediastinal region. Pain in shoulder and arm, Horner's syndrome and a dry cough are the prominent symptoms. The X-ray picture is that of a triangular shadow arising from the upper superior mediastinum. Bronchoscopy rarely aids in the diagnosis of tumor in this region. After a test dose of X-ray therapy, surgical exploration is indicated, but in most cases the tumors are inoperable when diagnosed. 'Primary tumors of the thoracic cage are characterized by pain and often present a palpable mass. Pain may be in arm, chest or abdomen. The chondromata and osteomata are potentially malignant and should be surgically removed. The malignant group, such as osteosarcomata and fibrosarcomata, usually show evidence of some bone destruction and the results of roentgen therapy and radical surgery have been disappointing. Therefore, as soon as a benign chest wall tumor is diagnosed it should be removed surgically. All patients with symptoms of persistent bronchial irritation or obstruction should early have the benefit of bronchoscopy. The vast majority of benign intrathoracic tumors are liable to malignant change. With the exception of the lymphoblastoma group, roentgen therapy is palliative and not curative. Often such therapy is prolonged until the case becomes inoperable. At some time during their growth most intrathoracic tumors can be removed. If the diagnosis of intrathoracic neoplasm has been made by X-ray or bronchoscopy, and lymphosarcoma and metastases have been ruled out, surgical exploration is at once indicated.—*The Diagnosis and Treatment of Primary Intrathoracic Tumors, F. S. Dolley & Capt. L. A. Brewer III, M.C., J. A. M. A., April 3, 1943, 121: 1130.*—(*G. L. L.*)

Anlagen and Rest Tumors of the Lung.—In the classification of lung tumors the greatest attention has been focused on the study of tumors of bronchogenic origin because of their relative importance and comparative frequency. But all lung tumors do not arise from the basal cells of mature tissue structure, hence the classification by Womack and Graham of some tumors of the lung as "mixed tumors" has had the salutary effect of emphasizing the embryological course of certain tumors and of carrying the reasonable implication that some of these are benign. Although many such tumors may be unidermal, the terms "anlagen" or "rest" tumors more fully describe them. To illustrate this, four tumors of the lung, manifesting distinct

evidences of origin either in anlagen or from more than one germinal layer are described. The first two tumors occurred in newborn, full-term infants who had survived seven and five days, respectively. These were of definite embryonal or anlagen origin, having developed during intrauterine life. In the first instance embryonal bronchial budding was perpetuated in a neoplastic mass. This was demonstrated by an aimless reduplication of tubular and immature alveolar structure indicative of a lack of orderly sequence of bud-branching. In the second case, evidence of both entodermal and mesodermal origin was revealed by areas of varied predominance of one or the other derivatives. The third tumor occurred in a thirty-four year old male who had suggestive signs of intrabronchial growth. Specimens were obtained through bronchoscopic removal in 1935 and again in 1939. The two specimens were evidently representative of mixed germinal layers. In the first a mesenchymal derivative was predominant while in the latter the tumor growth consisted to a great extent of elements of entodermal origin. It is interesting to note that this patient has remained well and is free from all former signs and symptoms. The fourth tumor occurred in a person nineteen years of age who had a history of occasional haemoptysis since the age of seven years. Signs and symptoms of a persistent intrabronchial growth with ball-valve action were present. Examination of a specimen obtained through the bronchoscope showed a neoplasm of mixed dermal components as revealed by the prevalence of both entodermal and mesenchymal elements. While the biological characteristics of malignancy and benignancy are more or less constant regardless of the primary mature or embryonal origin of neoplasms, there are precedents for a benign nature for many tumors having the latter origin. Such serve to promote conservatism in the evaluation of the relative malignancy of the pulmonary growths which may belong to this group. (With 8 plates.)—*Anlagen and Rest Tumors of the Lung Inclusive of "Mixed Tumors" (Womack and Graham), W. H. Harris & H. J. Schattenberg, Am. J. Path., September, 1942, 18: 955.*—(*J. S. W.*)

Roentgen Therapeutic Test Dose in Differential Diagnosis of Mediastinal Tumors.—By roentgen therapeutic test dose is meant the largest amount of given radiation that can be tolerated without producing injury to the normal structures. Its administration has a double aim: (*a*) to constitute a test of radiosensitivity when there are no other possibilities for differential diagnosis, and (*b*) to act as a therapeutic agent in case the tumor is radioresponsive. On the basis of the irradiation response of 73 cases of mediastinal tumors treated, it is possible to place the mediastinal tumors into four arbitrary groups. Group 1 includes tumors originating from the proliferation of the highly radiosensitive lymphocytic cell element, such as lymphocytoma; mediastinal manifestations of aleukaemic leukaemia, pseudoleukaemia and lymphatic leukaemia; and lymphosarcoma of the thymus. The regression of these tumors following the administration of the roentgen therapeutic test dose is spectacular. They completely disappear within two or three weeks after treatment. Three examples are cited for the purpose of illustration. Group 2 includes mediastinal tumors containing predominantly reticulo-endothelial or embryonal spindle-cell elements, such as mediastinal Hodgkin's disease, reticulum-cell sarcoma, endothelioma and embryonal spindle-cell sarcoma. The rate of regression of these tumors is moderate. After two weeks there is a 25 per cent reduction in size, after six weeks a 50 per cent or more reduction and after three months nearly always complete disappearance. In this group, often a second series of radiation therapy is given after the six weeks' period, as a rule with considerably less dosage. Most of the cases are Hodgkin's disease. Thirty-three per cent of these have survived the five year period. Those who died have only in exceptional instances shown a mediastinal recurrence. Not infrequently mediastinal Hodgkin's disease invades the anterior chest wall, producing external tumefaction, which,

especially in the presence of a positive serological test, is very difficult to differentiate from aneurysm. This occurred four times in the series. Group 3 includes other malignant tumors, such as fibrosarcoma, melanosarcoma, carcinoma of the thyroid and thymic gland, teratoma, etc. The rate of regression of these tumors is slow. After two weeks there is hardly any reduction in size, after six weeks there is a 20 to 50 per cent reduction and after three months in the majority of the cases there is again progression. In some instances, by administering further intensive irradiation, a notable prolongation of life or even five year survival is obtained. This group may be termed a group by exclusion. All tumors which cannot be fitted in the other groups are placed herein, with the notation that they represent malignant neoplasms of low radiosensitivity. But beyond that the application of the roentgen therapeutic test dose permits no further differentiation. Group 4 includes benign tumors of the mediastinum, such as neuroganglioma, dermoid cyst, chondroma, fibroma, lipoma, etc., and pseudotumors such as aneurysm, encysted fluid, encapsulated abscess, echinococcus cyst, etc. These tumors are uninfluenced by the administration of the roentgen therapeutic test dose, so that periodical checkup examinations reveal no change or very slow increase in size, compatible with the nature of the lesion. A possible exception is the very slow shrinkage of a large tumor believed to be a fibroma. In none of the 5 cases in this group in which the tumors were removed at intervals of from three months to ten years after the administration of the test dose was there the slightest difficulty encountered at the time of the surgical extirpation.—*The Value of the Roentgen Therapeutic Test Dose in Differential Diagnosis of Mediastinal Tumors, L. Reynolds & T. Leucutia, Am. J. Roentgenol., October, 1942, 48: 440.*—(*W. H. M.*)

Location and Extent of Pulmonary Embolism.—The high percentage of deaths from pulmonary embolism emphasizes the need for a clinical method of determining the size and position of the embolus. To study this experimentally, a solid embolus was first produced in the rabbit's lung, by injecting such embolus-producing substances as paraffin or paraffin mixed with lead filings directly into the right ventricle. Pulmonary embolism is determined from the roentgenogram by the presence of an infarct which does not develop until at least twenty-four hours have elapsed. The extent of the embolus is determined by the intravenous injection of diodrast which outlines the pulmonary arteries up to the occlusion. The diodrast is injected into a peripheral vein and six chest roentgenograms are taken within six to eight seconds following the injection, by the use of a specially constructed cassette-changer. The authors feel that the method employed should be applicable to postoperative embolism in man, since emboli large enough to cause serious damage can easily be identified by this means.—*The Use of Diodrast in Determining the Location and Extent of Pulmonary Embolism, F. Liberson & I. R. Liberson, Am. J. Roentgenol., September, 1942, 48: 352.*—(*W. H. M.*)

Mediastinal Emphysema.—The incidence of spontaneous pneumomediastinum or mediastinal emphysema is probably greater than previously supposed. Some of these cases have undoubtedly been diagnosed as coronary disease. Pathologically the process appears to begin with escape of air into the interstitial tissues of the lung from ruptured alveolar walls. The air passes along the connective tissue paths to the mediastinum where it collects around the vessel walls and between the heart and the anterior chest wall. It may also push out laterally, displacing one or both lungs. Hamman suggests that sometimes the air may work back from the mediastinum and reach the pleural cavity, thus producing a spontaneous pneumothorax. As the pressure increases there is frequently an infiltration of air up into the subcutaneous tissues of the neck. It is conceivable that the air may pass through the oesophageal hiatus or along the vessel walls through the diaphragm to infiltrate the retroperitoneal tissues. If the

mediastinal pressure becomes as great as that of the venous and intraauricular pressure, circulation ceases. The sudden onset of severe pain over the sternum or left chest is characteristic. On auscultation there is a peculiar crackling sound over the apex of the heart synchronous with the heart beat. On percussion there is diminished or absent cardiac dulness or even hyperresonance. A striking finding is subcutaneous emphysema of the neck. Occasionally pneumothorax is present. The temperature and pulse rate are normal, but respirations are shallow because of the pain. The blood count and electrocardiogram are normal. The roentgen findings are characteristic when present. In the posteroanterior view, there is a white line paralleling one or both borders of the cardiac shadow, which represents the border of the mediastinum as the lung is pushed out from behind the heart shadow. The lateral view shows a large air space between the heart and the anterior chest wall. The treatment is symptomatic with complete rest in bed to avoid the increased pain accompanying movement and exertion. Withdrawal of air may be indicated in certain cases. The author reports one case showing characteristic clinical and roentgenological findings in a young male adult.—*Spontaneous Pneumomediastinum, D. S. Kellogg, Am. J. Roentgenol., October, 1942, 48: 510.*—(*W. H. M.*)

Cystic Lung.—The authors present the following classification of lung cysts based on clinical, radiological and histological grounds.

Broncho-alveolar cysts (congenital or acquired):

(A): Alveolar cysts (air containing)
1. Pneumatocele or solitary alveolar cyst
   (a) Nonexpansile pneumatocele
   (b) Expansile or giant pneumatocele
2. Cystic emphysema or multiple alveolar cysts.
   (a) Diffuse cystic emphysema
   (b) Localized cystic emphysema

(B): Bronchial cysts (air or fluid containing)
1. Pneumocyst or solitary bronchial cyst
   (a) Nonexpansile pneumocyst
   (b) Expansile or giant pneumocyst
2. Cystic bronchiectasis or multiple bronchial cysts
   (a) Diffuse cystic bronchiectasis
   (b) Localized cystic bronchiectasis

   Both these varieties comprise:
   (1) Sponge type
   (2) Berry type
   (3) Honeycomb type

Case histories are included to illustrate the various types of cysts mentioned in the classification. Two theories are offered as to the manner of causation of air cysts: (*1*) The congenital defect theory which assumes that the terminal respiratory passages fail to reach proper maturity and form instead blind, nonrigid tube-like rests. Thus it is claimed that cysts can arise from the bronchi, bronchioles, atria or alveoli. (*2*) The acquired defect theory assumes that cystic formations arise as a result of acute and chronic inflammations which cause bronchial wall damage, fibrosis and emphysema. Most writers are agreed that both types of cysts are met. The main clinical features characterizing each type of cystic lung are tabulated with reference to the clinical history, symptoms, physical signs, radiological appearance, bronchographic, thoracoscopic and bronchoscopic findings as well as the histo-pathology.—*Cystic Disease of the Lung (Broncho-Alveolar Cysts), F. E. S. Willis & J. Almeyda, Tubercle, February & March, 1943, 24: 27, 43.*—(*E. H. R.*)

Broncholithiasis.—Bronchial stones usually originate within the pulmonary parenchyma and secondarily invade the bronchus. The cause of migration is infection and suppuration. Tuberculosis is by far the most common cause of stone formation within the lung,

but circumscribed pneumonias, abscesses, mycoses and infarcts occasionally are aetiological agents. As the stone approaches the bronchus, the symptoms of partial bronchial occlusion obtain, and they may be very severe with production of valvular stenosis. Later on, when occlusion is complete, atelectasis, sputum retention and suppuration occur. Migration of stones into the main bronchi has been observed bronchoscopically by means of repeated examinations, and several cases of this type are reported in the literature. A case of a fifty-four year old man is related in detail. The patient had a calcified primary focus in the lung, and calcified hilar lymph nodes. In addition, he had bronchiectasis in the adjoining pulmonary parenchyma. The infection caused the calcific focus in the hilar node to ulcerate into the bronchus with progressive symptoms over a period of two years. Finally the stone was removed bronchoscopically with complete symptomatic relief, and with disappearance of a considerable infiltration which had been observed on X-ray.—*Über Broncholithiasis; Entfernung eines Bronchialsteines auf endoskopischem Wege, F. Nager, Schweiz. med. Wchnschr., January 2, 1943, 73: 6.*—(*H. M.*)

Laryngotracheobronchitis in Children.—This is a true inflammatory disease extending from the throat down to the bronchi, and possibly to the bronchioles. Early, the airway is restricted by oedema and, later, necrosis and sloughing of the membrane and plugging of the bronchioles may develop with resultant atelectasis, emphysema and possibly pneumonia. The majority of cases occur during the first two years of life. The beta-haemolytic streptococcus, staphylococcus aureus, haemophilus influenzae as well as pneumococci are the offending organisms. Treatment involves teamwork between the physicians, the nursing staff and the laboratory. A room containing a cold humidifier in which the temperature is kept at approximately 70 to 75°F. is considered superior to a hot steam room. If possible, throat, tracheal and blood cultures are obtained. A tracheal tube and oxygen may be very helpful and if necessary a tracheotomy should be performed. Catheterization of the bronchi and aspiration of the pus is easily possible after the tracheotomy. Full doses of sulfonamides or penicillin should be started early. Transfusions may be useful. Morphine and atropine are contraindicated. Ipecac is not needed. Fluids should be given in moderate amounts by easiest available route.—*Acute Laryngotracheobronchitis, J. A. Davies, New England J. Med., July 29, 1943, 229: 197.*—(*J. B.*)

Bronchial Obstruction.—A sixteen year old white female was admitted to the Groote Schuur Hospital with signs and symptoms of bronchiectasis and pneumonia in the right lower lobe. On bronchoscopy an obstruction with a polypoid appearance was found in the right bronchus. Biopsy showed thyroid tissue. The entire growth was removed.—*A Case of Bronchial Obstruction, P. W. J. Keet, Clinical Proceedings, Cape Town, April, 1943, 2: 119.*—(*G. C. L.*)

Broncho-oesophageal Fistula. — A thirty-eight year old colored female was admitted to the Groote Schuur Hospital with the following symptoms: cough, dysphagia, substernal pain, vomiting and paroxysmal cough occurring as soon as she swallowed food. Radiological examination showed a broncho-oesophageal fistula. A biopsy from the oesophagus revealed inflammatory tissue. The diagnosis of a syphilitic ulcer originating in the oesophagus was made. The fistula disappeared after antiluetic treatment.—*A Case of Broncho-Oesophageal Fistula, P. W. J. Keet, Clinical Proceedings, Cape Town, April, 1943, 2: 118.*—(*G. C. L.*)

Diaphragmatic Hernia.—An extensive herniation into the left pleural cavity was seen in a stillborn female infant of colored stock. The contents of the hernia were: the stomach, the small intestine, the caecum, the appendix, the ascending colon, the transverse colon, half of the descending colon, part of the liver, the spleen, almost the whole pancreas. No

hernial sac was present. Only two narrow strips of muscles of the left hemidiaphragm were found. This hernia belonged to the pleuro-peritoneal group which can be distinguished from the hiatal hernia. It is caused by a failure of the left pleuro-peritoneal fold to grow and form part of the diaphragm. —*A Case of Extensive Diaphragmatic Hernia in a Stillborn Infant, J. A. Keen, Clinical Proceedings, Cape Town, February, 1943, 2: 44.*—(*G. C. L.*)

Hiatus Hernia.—The clinical picture of a hiatus hernia is of sufficient frequency to be included in the differential diagnosis of anterior chest or upper abdominal complaints. It may simulate coronary artery disease, oesophagospasm, peptic ulcer or gall bladder disease. The manifestations are so variable and inconstant that clinical diagnosis is usually impossible without the aid of detailed and careful roentgenographic examination of the abnormality. A symptom which occurs in relatively few cases but which is pathognomonic is the feeling of distress or pain or fulness in the epigastrium during or soon after eating, being relieved by walking about for a short time. Gastrointestinal bleeding is the most important complication of the disease. The treatment is essentially medical, consisting of a bland diet divided into four or six feedings. The patient is to remain in the upright position after a meal. Alkalies and antispasmodics are useful. Surgery is indicated when medical measures fail to give relief.—*Diaphragmatic (Hiatus) Hernia, W. R. Ohler & M. Ritus, New England J. Med., July 29, 1943, 229: 191.*—(*J. B.*)

Sensitivity to Tuberculin in Acne.—It has been noted that acne is a frequent disease of the skin among inmates of tuberculosis sanatoria and this has been ascribed to the average youthful age of the inmates or possibly to the fattening diet employed. That there is any direct relation between acne and tuberculosis has never been demonstrated. The present paper offers a comparison of the sensitivity to tuberculin of 102 persons with acne and 129 persons with other nontuberculous cutaneous diseases, the latter serving as controls. While in the age group from thirteen through twenty-five years the proportion of positive reactors among the subjects with acne and the controls was practically the same, in the age group from twenty-six through forty years there was a decidedly smaller proportion of positive reactors among those with acne than among the controls. A further analysis of the material showed that patients with severe acne tended to show negative to weakly positive reactions to tuberculin and that strongly positive reactions were associated with mild acne. There appears to be a relation between the degree of sensitivity of the skin to tuberculin and (*a*) acne as it occurs in older age groups or (*b*) the severity of the acne. The nature of this relation, however, is not elucidated.—*Sensitivity to Tuberculin in Acne and in Other Nontuberculous Diseases of the Skin, K. Loewenthal, Arch. Dermat. & Syph., June, 1943, 47: 799.*—(*J. S. W.*)

Chest Wall Infections.—Bülau recommended closed intrapleural drainage for empyema in 1870. Since then it has been used extensively with good results, and it still is the only practicable method for treating empyemata in the field under war conditions. Phlegmonous infections of the chest wall occurring during the course of treatment are occasional complications and immediately warrant a very serious prognosis. The frequency of this complication is given variously between 1 and 3 per cent. In the university clinic in Basel, 4 cases were seen in the years 1938 to 1941, 3 of which proved fatal. Once an infection has developed, the treatment consists of wide incisions reaching into healthy tissue. Powdered sulfa drugs may be placed into the wounds and thoracotomy for drainage is done at the same time. To avoid this serious complication, the treatment of empyemata occurring late in the course of pneumonia with rib resection and open drainage is advised. Early empyema cannot be treated in this fashion, and more recently closed drainage has been performed by trephining a

hole into a rib and placing the catheter through this. The incidence of infections can be lowered by observing a few simple rules. The tube should be placed at the lowest point of the empyema cavity, and preferably between the posterior axillary and scapular lines. The skin and muscles should be incised for at least 3 cm., and the trocar should be 6 to 7 mm. in thickness. The catheter is inserted not more than 2 cm. into the pleural cavity and secured firmly. In putrid empyemas, the injection of gas gangrene antiserum into the surrounding tissues is advised. The wound is left open and packed with antiseptic gauze, and the dressing should be airtight.—*Brustwandphlegmone nach Bülau-Drainage des Pleuraempyems, L. Jeker, Schweiz. med. Wchnschr., April 3, 1943, 73: 415.*—(*H. M.*)

Cardiac Arrhythmias following Pneumonectomy.—A study is reported of the incidence of cardiac arrhythmias, especially functional auricular fibrillation and flutter, following pneumonectomy. In a series of 78 post-total-pneumonectomy patients, there were 4 cases of auricular fibrillation, 2 cases of auricular flutter, one case of flutter-fibrillation and one case of nodal tacchycardia. One case developed fibrillation following a right middle and lower lobectomy. The patients' ages varied from thirty-two to sixty-six years. A careful history and physical examination failed to reveal evidence of heart disease in any case before operation. Five of these patients with postoperative arrhythmias had had a normal preoperative electrocardiogram. The arrhythmias usually appeared during the first two postoperative weeks although in one case fibrillation set in on the fifty-seventh day. In 8 cases, normal sinus rhythm was restored within forty-eight hours by rapid digitalization. No patient had developed cardiac decompensation or had had a return of the cardiac arrhythmias in a follow-up study. In a search for precipitating factors that initiated these arrhythmias it was found that none occurred in 63 cases following single lobectomy whereas 8 of 78 pneumonectomy cases developed the arrhythmias. This suggested that the more extensive procedure is essential to its production. The decided contralateral shift of the mediastinum following total pneumonectomy and the ensuing pleural effusion may play a part in initiating arrhythmias. Vagal stimulation caused by irritation of the nerve by a stitch abscess at the bronchial stump may be a precipitating factor inducing fibrillation. The aetiology is, however, unknown.—*Cardiac Arrhythmias following Pneumonectomy, C. C. Bailey & R. H. Betts, New England J. Med., August 26, 1943, 229: 356.*—(*J. B.*)

Key to Abstractors

E. H. R.: Eli H. Rubin, New York, New York.
G. C. L.: George C. Leiner, New York, New York.
G. L. L.: George L. Leslie, Fort Jackson, South Carolina.
H. M.: Henriette Marcus, New York, New York.
J. B.: Joseph Ballinger, New York, New York.
J. S. W.: J. Stanley Woolley, Otisville, New York.
W. H. M.: W. H. Merrit, New York, New York.